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AND HIV-RELATED MORTALITY

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The Link Between Public and Private Insurance and HIV-Related Mortality
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ABSTRACT

As policymakers consider expanding insurance coverage for HIV+ individuals, it is useful to ask if insurance has any effect on health outcomes; and, if so, whether public insurance is as efficacious as private insurance in preventing premature deaths among HIV+ patients. Using data from a nationally representative cohort of HIV-infected persons receiving regular medical care, we estimate the impact of different types of insurance on mortality in this population.

We find that ignoring observed and unobserved health status leads one to conclude (misleadingly) that insurance may not be protective for HIV patients. After accounting for observed and unobserved heterogeneity, insurance does protect against premature death, but private insurance is more effective than public coverage. The better outcomes associated with private insurance are attributable to the more restrictive prescription drug policies of Medicaid.

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1 Introduction

Most economic models treat health insurance as a hedge against financial risk from illness. However the benefits from health insurance are not limited to the avoidance of financial risk, additional benefits are derived from insurance's ability to make available medical care that would otherwise be unaffordable (Nyman, 1999). In fact, policymakers have often cited the lack of affordability of health care and consequent poor health outcomes for the uninsured as the primary reason for expanding health insurance coverage. This view has motivated many investigations of the link between health insurance and health, including a recent study by the Institute of Medicine (Institute of Medicine, 2002).

In their review of this literature, Levy and Meltzer (2001) found that most of these studies were flawed. There was no causal link between having health insurance and better health—rather, there were associations. Exceptional studies that use randomization or quasi-randomization to address causation have found either no or limited effects on health, depending on the population under investigation and the health measure used. One problem with asking the broader question of how insurance affect health is that it may just be too ambitious. The impact of insurance on health will obviously depend on circumstances; from a policy perspective, it might be more useful to ask what population can most benefit from insurance expansions (either public or private), and how this relationship changes with changes in medical treatment.

The treatment of human immunodeficiency virus (HIV) provides a useful case study. In 1995, the Food and Drug Administration approved the first protease inhibitor for treatment of HIV. When new viral particles break off from an infected cell, protease cuts long protein strands into the parts needed to assemble a mature virus. These drugs block the protease enzyme, and hence prevent new viral particles from maturing. Clinical trials and observational data soon confirmed their clinical usefulness in combination with older drugs (so-called highly active anti-retroviral therapy or HAART) in forestalling HIV-related mortality (Palella et al., 1998). After the introduction of protease inhibitors into clinical practice, age-adjusted death rates from HIV infection fell 25% from 1995 to 1996 and 47% from 1996 to 1997 (Center for Disease Control, 1998).¹

¹HAART is a combination therapy involving three types of drugs: nucleoside reverse

HAART is expensive, costing on average about \$13,000 per year. Thus it is possible that health outcomes for HIV-infected persons will be very responsive to the availability of insurance. Indeed, in previous work, we found that insurance significantly reduced mortality in HIV+ patients (Goldman et al., 2001). These results suggest that policies to expand insurance coverage to the uninsured HIV+ population could save many lives. However, that paper treats insurance as a single category for computational ease. HIV+ patients have coverage either through private insurance or public insurance—typically Medicaid. Since these types of insurance differ in extent of coverage and provider reimbursement we would expect that they differ in the quality and intensity of treatment.

Several studies have examined the relationship between insurance type and service utilization among HIV+ patients (Fleishman et al., 1994; Horner et al., 1996; Joyce et al., 1998; Shapiro et al., 1999). Most of these studies find that the uninsured or publicly insured HIV+ patients incur lower per diem charges, receive fewer procedures and are less likely to receive expensive drug therapy than privately insured HIV+ patients with similar medical conditions. In addition, a majority of HIV patients covered by public insurance are required to demonstrate a disability to qualify for coverage. Thus, most patients with public insurance obtain coverage only in the advanced stage of disease. These differences in the intensity of treatment and timing of initiation of coverage among publicly and privately insured HIV+ patients leads naturally to questions about how these insurance types differ in their efficacy in preventing premature HIV related deaths.

In this paper, we identify the causal effects of different types of insurance on mortality by analyzing observational data on a nationally representative sample of adult HIV+ patients receiving care in the US. However, measuring the efficacy of insurance in observational data is complicated by the fact that insurance decisions might be correlated with unobserved differences in the health status of patients. Thus, to account for this endogeneity of insurance choices we jointly estimate insurance decisions and mortality and allow for arbitrary correlation between insurance and mortality in a parametric setting. To identify the ‘true’ effect of different insurance types on mortality we use state policy variables that affect the ease with which patients’ can ob-

transcriptase inhibitors, protease inhibitors, and non-nucleoside reverse transcriptase inhibitors. All regimens require at least three drugs, and the vast majority involve at least one protease inhibitor.

tain public insurance and a state level variables for firm size that affects the likelihood of obtaining employer provided insurance as instruments. These variables are clearly related to patient insurance status, but should not directly affect death rates (except through insurance).

The next section describes the data used for this analysis. We then present our joint model of insurance and mortality and provide the details of our identification strategy. Following that, we present results from a reduced form model that treats insurance as exogenous and compare them to the results from our structural model. The final sections discuss the conclusions and limitations of our analysis.

2 Data

We use data from a nationally representative study of HIV+ patients in care—the HIV Costs and Services Utilization Study (HCSUS). The HCSUS employed a multi-stage national probability sample design to identify HIV+ patients over 18 years old, who made at least one visit for regular care in the contiguous United States in January or February of 1996. It does not include HIV+ patients whose only contact with the health care system was through military, prison, or emergency department facilities, or who have not made contact with the health care system for their HIV. HCSUS collected three rounds of interviews: baseline, a first follow-up and a second follow-up with a baseline response rate of 68% (Duan et al., 1999). The first follow-up interview was conducted with 2466 subjects between December 1996 and July 1997; and the second follow-up was conducted with 2267 subjects between August 1997 and January 1998. Much of the attrition between waves is due to mortality. Toward the end of data collection for the baseline survey, HAART entered clinical practice and disseminated widely as the first-follow-up survey was in the field. Thus, we use the first follow-up sample of 2466 subjects with covariates measured at first follow-up for our analysis.

We construct analytic weights to adjust the sample to the reference population. A respondent’s analytic weight, which may be interpreted as the number of people in the population represented by that respondent, is the product of three patient-specific quantities – the sampling weight, the multiplicity weight, and the non-response weight. The sampling weight adjusts for oversampling (of women, for example); the multiplicity weight adjusts for patients who could potentially enter the sample via multiple providers;

and the non-response weight adjusts for differential cooperation (Duan et al., 1999). All analyses presented in this paper use these weights.

We use mortality one year after the interview date as our outcome variable. Mortality is based on data collected by HCSUS interviewers in the field, as well as from Equifax, Inc., a credit company that also tracks deaths in the United States. The main explanatory variable we are interested in is insurance type, which are derived from the HCSUS data. Table 1 shows the proportion of respondents on HAART by sources of insurance coverage. Table 1 shows that the majority of HIV+ patients obtain coverage through either private insurance or Medicaid – 30% of the respondents are covered by private insurance only, 28% are covered by Medicaid only, 16% are covered by both Medicaid and Medicare, and 18% are uninsured. However there are sharp differences in the use of HAART by source of insurance coverage. HIV patients with private insurance only are more likely to be on HAART than patients with any public insurance coverage. This is despite the higher incidence of AIDS (more advanced disease) among patients with public insurance. Thus to account for this differential use of HAART we classify patients into three mutually exclusive insurance categories –private insurance only, (any) public insurance, no insurance.²

3 Empirical Model of Insurance and Mortality

Let m_i^* represent an index function that measures the mortality propensity for HIV+ patient i one year after interview. We model this mortality propensity as a function of the insurance status of the individual, observed covariates, and and unobserved error:

$$m_i^* = c_1 + \gamma \times insured_i + \beta_1' X_i - \varepsilon_i \quad (1)$$

The vector X_i represents observed exogenous covariates that determine mortality propensity, such as age, gender, and education. Mortality is also affected by insurance status, where $private_i$ represents whether the patient was covered by private insurance (only), and $public_i$ represents whether the patient was covered by (any) public insurance. Mortality is also assumed

²We check the sensitivity of our results in an alternate specification where we exclude patients with Medicaid and private insurance, Medicare and private insurance, and Medicare only from the public insurance category as patients in these insurance categories had higher use of HAART than other patients in the public insurance category.

to depend on an unobservable heterogeneity component, $\rho_{m,i}$ that will also relate to insurance choices. It is useful to think of this as unobserved health status or attitudes towards risk, and they are assumed to be orthogonal to the covariates X_i . There is also a random error $\varepsilon_{m,i}$ that is uncorrelated with X_i and insurance choice. We want to consistently estimate the parameters c_1 , β_1 , γ_1 and γ_2 , after accounting for the endogeneity of insurance status.

We define m_i as an indicator variable that represents whether patient i actually died one year after interview:

$$m_i = \begin{cases} 1 & \text{if } m_i^* > 0 \\ 0 & \text{if } m_i^* \leq 0 \end{cases} \quad (2)$$

We assume that $\varepsilon_{m,i}$ are i.i.d standard normal errors, with zero mean and unit variance. This distributional assumption implies a probit model for m_i , where the probability of death, conditional on observed characteristics $\{private_i, public_i, X_i\}$ and unobserved characteristics ρ_m is:

$$P[m_i = 1 | \{private_i, public_i, X_i\}, \rho_m] = \Phi(c_1 + \gamma_1 private_i + \gamma_2 public_i + \beta_1' X_i + \rho_{m,i}) \quad (3)$$

Here, $\Phi(\cdot)$ is the cumulative distribution function for the standard normal distribution.

Patients choose among insurance types $j = \{private, public, uninsured\}$ on the basis of a standard random indirect utility function:

$$V_{j,i}^* = c_j + \beta_j' Z_i + \rho_{j,i} + \varepsilon_{j,i} \quad (4)$$

Here, Z_i represents variables that determine insurance status including our set of instrumental variables (that is, variables that belong in the insurance equation, but not in the mortality equation); and $\rho_{j,i}$ is a patient-specific random intercept that reflects the patients' propensity for insurance status j that is unobserved by the researcher. Again, this could be due to unobserved disease severity or preferences for risk. The parameters c_j and β_j are additional parameters to be estimated; and $\varepsilon_{j,i}$ represents the orthogonal error term.

Patients choose the insurance status that maximizes their indirect utility. We assume that $\varepsilon_{j,i}$ are independently and identically distributed according to the Type II extreme value distribution. This distributional assumption and normalizing $\{c_{uninsured}, \beta_{uninsured}, \rho_{uninsured,i}\}$ to zero yields a multinomial logit model for insurance choice.

$$P[\textit{private}_i = 1 | Z_i, \rho_{\textit{private}}, \rho_{\textit{public}}] = \frac{\exp(c_{\textit{private}} + \beta'_{\textit{private}} Z_i + \rho_{\textit{private},i})}{1 + \sum_{j \neq \textit{uninsured}} \exp(c_j + \beta'_j Z_i + \rho_{j,i})} \quad (5)$$

$$P[\textit{public}_i = 1 | Z_i, \rho_{\textit{private}}, \rho_{\textit{public}}] = \frac{\exp(c_{\textit{public}} + \beta'_{\textit{public}} Z_i + \rho_{\textit{public},i})}{1 + \sum_{j \neq \textit{uninsured}} \exp(c_j + \beta'_j Z_i + \rho_{j,i})} \quad (6)$$

To complete the model and allow for correlation between mortality and insurance choices, we need to assume a joint distribution for the unobserved heterogeneity vector $\rho = (\rho_m, \rho_{\textit{private}}, \rho_{\textit{public}})$. Our approach is semi parametric. We allow the unobserved heterogeneity in each equation to take one of three values—intuitively, there are three types of people that occur with probabilities p_1 , p_2 , and $1 - p_1 - p_2$. The effect of being a certain type has different effects on each outcome: $(\rho_m^1, \rho_m^2, \rho_m^3)$ for mortality, $(\rho_{\textit{private}}^1, \rho_{\textit{private}}^2, \rho_{\textit{private}}^3)$ for private insurance, and $(\rho_{\textit{public}}^1, \rho_{\textit{public}}^2, \rho_{\textit{public}}^3)$ for public insurance. For example, there is a p_1 probability that a person will be of the first type, which would imply realizations of ρ_m^1 for the propensity to die, $\rho_{\textit{public}}^1$ for the propensity to have public insurance, and $\rho_{\textit{private}}^1$ for the propensity to privately insurance.

This discrete factor distributional approach has several advantages over specifying a continuous parametric density for the unobserved heterogeneity vector. First, an incorrect specification of the parametric density function might lead to biased parameter estimates. The discrete factor density allows us to approximate any underlying distribution of heterogeneity. In fact, Monte Carlo studies show that discrete factor distributions with 2-4 points of support adequately model many distributions (Heckman, 2001; Mroz, 1999). Second, discrete factor models are computationally simpler than parametric models as they avoid multiple numerical integration in the construction of the likelihood function.

Since the mortality, public insurance, and private insurance equations have intercept terms, we normalize the mean of each heterogeneity component to be zero. This implies that the third point of support in each equation is not “free”.³ Thus we need to estimate 8 additional parameters: 2 points of

³For example, the third point of support ρ_m^3 in the mortality equation can be written as

support in the mortality equation, 2 points of support in the private insurance equation, 2 points of support in the public equation, and 2 probabilities. The resulting variance-covariance matrix for the unobserved heterogeneity is:

$$Var(\rho_m, \rho_{private}, \rho_{public}) = \begin{bmatrix} \sum_k p_k (\rho_m^k)^2 & \sum_k p_k \rho_m^k \rho_{private}^k & \sum_k p_k \rho_m^k \rho_{public}^k \\ & \sum_k p_k (\rho_{private}^k)^2 & \sum_k p_k \rho_{private}^k \rho_{public}^k \\ & & \sum_k p_k (\rho_{public}^k)^2 \end{bmatrix} \quad (7)$$

This model not only allows non-zero covariance across mortality and insurance propensities but also allows non-zero covariance between private and public insurance propensities. Thus our model relaxes the IIA assumption of standard multinomial logit model and allows completely general variance-covariance matrix. The key correlations in our model may thus be written as:

$$Corr(\rho_m, \rho_{private}) = \frac{\sum_k p_k \rho_m^k \rho_{private}^k}{\sqrt{\sum_k p_k (\rho_m^k)^2 \sum_k p_k (\rho_{private}^k)^2}} \quad (8)$$

$$Corr(\rho_m, \rho_{public}) = \frac{\sum_k p_k \rho_m^k \rho_{public}^k}{\sqrt{\sum_k p_k (\rho_m^k)^2 \sum_k p_k (\rho_{public}^k)^2}} \quad (9)$$

$$Corr(\rho_{private}, \rho_{public}) = \frac{\sum_k p_k \rho_{private}^k \rho_{public}^k}{\sqrt{\sum_k p_k (\rho_{private}^k)^2 \sum_k p_k (\rho_{public}^k)^2}} \quad (10)$$

We use maximum likelihood to estimate the parameters of our model. The problem is that we do not observe any patient's type and so we must integrate over all possible types (three in our model). However, conditional

a function of the other two points of support and the probabilities of each type, as follows:

$$\begin{aligned} E(\rho_m) &= 0 \\ \Rightarrow p_1 \cdot \rho_m^1 + p_2 \cdot \rho_m^2 + (1 - p_1 - p_2) \cdot \rho_m^3 &= 0 \\ \Rightarrow \rho_m^3 &= -\frac{p_1 \cdot \rho_m^1 + p_2 \cdot \rho_m^2}{(1 - p_1 - p_2)} \end{aligned}$$

on a given type, the heterogeneity is known. The contribution of patient i to the likelihood function is thus given by:

$$\begin{aligned}
l_i = & \sum_{k=1}^3 p_k \left(\text{P} [m_i = 1 | \rho_m^k]^{m_i} \right) \times \left(1 - \text{P} [m_i = 1 | \rho_m^k]^{1-m_i} \right) \times \\
& \left(\text{P} [private_i = 1 | \rho_{private}^k]^{private_i} \right) \times \left(\text{P} [public_i = 1 | \rho_{private}^k]^{public_i} \right) \times \\
& \left(1 - \text{P} [private_i = 1 | \rho_{private}^k] - \text{P} [public_i = 1 | \rho_{private}^k] \right)^{(1-private_i)(1-public_i)}
\end{aligned} \tag{11}$$

We have 6 possible outcomes for the dependent variables in our sample: (dead/alive) x (none/private/public). To construct the contribution to the likelihood function for each set of outcomes, we first obtain the likelihood of observing that value of the dependent variables conditional on a realization k of the unobserved heterogeneity $\rho^k = (\rho_m^k, \rho_{private}^k, \rho_{public}^k)$. We then integrate over all all possible realizations to get Equation (11). Finally we obtain the weighted log-likelihood function by summing the log-likelihood across individuals:

$$\ln(L|\Gamma) = \sum_{i=1}^N w_i \ln(l_i) \tag{12}$$

Γ is the vector of model parameters; w_i are the analytic weights and N is the sample size. Because it is difficult to interpret the magnitude of the parameter estimates directly, we also report the relative impact of private and public insurance on average mortality. More precisely, we compute:

$$\begin{aligned}
\text{RI (private)} & \equiv \frac{\text{Average mortality private}}{\text{Average mortality not insured}} - 1 \\
& = \frac{\sum_i \sum_k p_k \Phi(c_1 + \gamma_1 + \beta'_1 X_i + \rho_m^k)}{\sum_i \sum_k p_k \Phi(c_1 + \beta'_1 X_i + \rho_m^k)} - 1
\end{aligned} \tag{13}$$

$$\begin{aligned}
\text{RI (public)} & \equiv \frac{\text{Average mortality public}}{\text{Average mortality not insured}} - 1 \\
& = \frac{\sum_i \sum_k p_k \Phi(c_1 + \gamma_1 + \beta'_1 X_i + \rho_m^k)}{\sum_i \sum_k p_k \Phi(c_1 + \beta'_1 X_i + \rho_m^k)} - 1
\end{aligned} \tag{14}$$

4 Identification

We use state Medicaid policies and average firm size as our instrumental variables to explain insurance status but not mortality (except via insurance status). Medicaid is the most common form of insurance for the HIV+ population in care, covering 46% of the population. HIV+ patients can qualify for Medicaid through three distinct pathways.

First, patients who meet the state’s income eligibility and family composition requirements for Aid to Families with Dependent Children (AFDC) as they existed on July 16, 1996 qualify for Medicaid coverage. Second, Supplemental Security Income (SSI) beneficiaries are automatically eligible for Medicaid in 38 states. The other states have different standards for eligibility either as a 209(b) state or a waiver state. Section 209(b) of the Social Security Amendments Act of 1972 allows States to include more restrictive definitions of “disability” and lower income and assets standards for Medicaid eligibility. Medicaid eligibility is also available through a “medically needy” program for individuals who meet Medicaid’s disability criteria but have incomes that exceed the financial eligibility limit. The program allows individuals to “spend down” to Medicaid eligibility by deducting medical-related expenses from their reported income. States have the option to but are not required to establish a “medically needy” program. In addition, states vary in their income eligibility levels for the “medically needy” program. For each patient, we define the following four variables based on Medicaid eligibility rules in the state in which the patient is sampled:

- *Medically-Needy Threshold*: This is the state’s income eligibility threshold for the medically-needy program expressed as a percentage of the federal poverty line⁴.
- *AFDC Threshold*: This is the State’s income eligibility threshold for Aid to Families with Dependent Children (AFDC) eligibility in 1996 expressed as a percentage of the federal poverty line.
- *SSI < 65% FPL (federal poverty line)*: This is an indicator variable for whether the state’s income eligibility threshold for Medicaid eligibility

⁴States which did not institute a medically-needy program were coded as having an income threshold of zero percent

through the “SSI” category was at least 10 percentage points lower than the federal guideline of 75 percent of the federal poverty line⁵.

- *Average firm size*: This is the average firm size in the state in which the patient was sampled as instrument for insurance status. Several studies have documented the strong positive association between firm size and employer provided insurance offers (Bundorf, 2002; Cantor et al., 1995; Employee Benefit Research Institute, 2001).

Distributional assumptions aside, as in all instrumental variable-based studies, the credibility of our study rests on the believability of our instruments. Our state policy instruments and average firm could fail in at least two ways. First, the estimators perform poorly if the instruments are only weakly correlated with the treatment variable—that is, insurance status (Nelson and Startz, 1990; Bound et al., 1995; Staiger and Stock, 1997). Thus, we report Wald statistics for the joint significance of our instruments in predicting insurance status. Second, our instruments might be correlated with unobserved determinants of mortality (like unmeasured health status variables). The assumption that an instrumental variable is uncorrelated with the outcome measure cannot be directly tested. For these reasons, some researchers have argued that IV estimates in this context should be viewed with caution (Bound et al., 1995).

However, in our application, it seems clear that patients have little direct influence at an individual level on state policies or firm size, so our state level instruments seem valid. This argument is not enough to establish exogeneity, however, if there are unobserved state-level variables that determine both health and insurance status. In that case, state policies would be endogenous in our model despite the lack of control by patients over these policies. In order to address this issue, we develop some indirect evidence that our instruments are not simply picking up differences in unobserved health across states. If the latter hypothesis were true, then one would expect to find that our state-level instruments predict the mortality of patients even in a non-HIV population.

To check this assumption, we estimate a logit model of one-year mortality using data from the Medicare Current Beneficiary Survey (MCBS). On the

⁵We coded this as a dummy variable as there were only a 4 states that implemented a significantly more restrictive eligibility standard than the federal guideline.

right-hand side, this mortality model includes a sparse set of health status indicators, such as measures of Activities of Daily Living (ADLs) and a general health index, and our state-policy instruments. Since this elderly or disabled population is by definition insured by Medicare, our instruments should not predict their mortality unless they proxy for unobserved state-level effects. Table 2 reports the regression results and shows that our instruments are not statistically significant in the model, with odds ratios near one. Of course, these results do not prove that unobserved state effects are unimportant in the HCSUS population, but they are certainly suggestive. There is no good reason to expect that such effects should be present for HIV+ patients when they are not present for the elderly or disabled.

5 Results

Descriptive means for all model variables are given in Table 3. Most of the variables are self-explanatory. In some models, we include measures of the lowest ever CD4+ t-lymphocyte cell count, a critical measure of the function of a patient’s immune system. A depletion in these cells correlates strongly with the worsening of HIV disease and the risk of death (Fauci et al., 1998). In this paper, we categorize CD4+ counts into four categories, as shown in Table 3. Patients with CD4+ lymphocyte counts below 50 have a very poor prognosis in general; while those with counts above 500 are considered much healthier.

To illustrate the consequences of selection bias, we estimate a “naïve” probit model where insurance status is treated as an exogenous variable. Table 4 reports the results from the naïve models. In order to demonstrate the importance of including information on health status, we include two sets of estimates; one set with controls for disease progression, particularly CD4 cell count, and one without.

In the regression without severity controls, public insurance is associated with an increased probability of 1-year mortality, and the effect is statistically and substantively significant. This finding persists in the analysis with severity controls, although the effect is no longer significant. This finding is analogous to that of Lancaster and Intrator (1998) who also found the perverse result that health insurance increases the risk of death for HIV+ patients. In both the regression models, private insurance is associated with a decreased probability of death, however the effect size is statistically in-

significant in both models.

Table 5 summarizes the results on the impact of insurance status on mortality. In general, the use of severity controls reduces the magnitude of the both public and private insurance effect substantially. For instance, the results without severity controls indicate that public insurance increases relative mortality by 168%, including health status measures like CD4 cell count reduces this effect to 31%, but it does not disappear. Similar results are obtained for the private insurance effect—private insurance decreases relative mortality by 9% without severity controls and by 46% with severity controls. We attribute these findings to a spurious correlation between severity of illness and insurance for HIV patients. The correlation between public insurance and severity of illness seems to be especially strong. A possible explanation is that the eligibility rules for Medicaid or Medicare require HIV+ patients to demonstrate a “disability” – almost always associated with advanced disease.

The parameter estimates for the mortality equation (1) in the structural model are shown in Table 6, along with the correlation between insurance status and mortality—equations (8) and (9)—and the correlation between public and private insurance—equation (10). Table 7 gives the parameter estimates for the private and public insurance equations and also reports the joint significance of the excluded instruments in equation (4). This is computed using a Wald statistic, since these are considered a test of the small sample bias associated with the IV estimator (Staiger and Stock, 1997).

Table 6 shows several important differences from the naïve results. First, we see that both private and public insurance decrease the likelihood of death. This is in sharp contrast to the results from the naïve model where public insurance was associated with a higher probability of death and private insurance had a statistically insignificant effect on mortality. This reversal in the effects of private and public insurance on mortality can be explained by the positive correlation between unobserved health status and insurance choice. For instance, our results show that unobserved mortality propensity and public insurance propensity are positively correlated (correlation coefficient 0.89). Similarly we also find positive correlation between unobserved mortality propensity and private insurance propensity, although the degree of correlation is much weaker than that for public insurance.

Second, the magnitude of the relative impact of insurance on mortality is greater for private insurance (79%) than for public insurance (66%) (shown in Table 5). These findings might be explained by the differential access to

HAART among those with public and private insurance (shown in Table 1). The findings are also consistent with the fact that most respondents receive public insurance only when they demonstrate “disability” due to advanced disease -this might delay treatment for the publicly insured patients thus resulting in poorer outcomes than those for privately insured patients.

In an alternate model we excluded patients with Medicare and private insurance, Medicaid and private insurance and Medicare only from the public insurance categories. Since these patients had higher HAART use than others on public insurance we would expect that excluding these patients would increase the difference between the impact of public and private insurance on mortality. Our results, confirm this hypothesis—the relative impact of private insurance on mortality increases marginally to 80% and the relative impact of public insurance on mortality decreases to 63%⁶.

It is also interesting to note that private and public insurance propensities are positively correlated. This might be explained by unobserved characteristics such as risk aversion and poor health that increase the propensity for insurance, in general.

The results from the insurance equations in Table 7 show that the state level instruments are highly correlated with both private and public insurance choice and are jointly significant ($p < 0.01$). As expected, we find that higher average firm size is associated with higher likelihood of having private insurance. We also find that more generous Medicaid eligibility rules are associated with higher likelihood of public insurance coverage. However, contrary to our expectations the results indicate that higher AFDC threshold is associated with a lower likelihood of public insurance. Multi-collinearity between the three Medicaid policy variables, which reduces the power of our estimates, seems to be the most likely explanation for this result (correlation between AFDC threshold and Medically Needy threshold is 0.62). In an alternate specification in which we excluded other Medicaid policy variables, we find that AFDC threshold has the expected sign and higher AFDC threshold is associated with higher likelihood of public insurance.

6 Conclusion

Our main findings are that (1) ignoring observed and unobserved health status in our structural mortality equation leads one to conclude that insurance

⁶The full results from this model are available from the authors upon request.

may not be protective for HIV patients, (2) after accounting for observed and unobserved heterogeneity, insurance does protect against death, and (3) private insurance is more effective than public insurance in protecting HIV patients.

The finding that insurance is protective surely is related to highly active anti-retroviral therapy. By July 1997, approximately 75% of the HCSUS sample had used a protease inhibitor or nonnucleoside reverse transcriptase inhibitor (Shapiro et al., 1999). This rapid proliferation in clinical practice is remarkable given the approval of the first protease inhibitor was only two years earlier, and the expense associated with these new drugs.

The finding that private insurance is more effective than public insurance in preventing premature HIV-related deaths might be explained by differences in the intensity of treatment and use of HAART. In the HCSUS, less than 5% of those with private insurance did not have drug coverage. But while all Medicaid programs provide prescription drug coverage to those who are categorically eligible, many states do not provide such coverage to the Medically-Needy⁷. Using program participation and income data in the HCSUS, we have estimated elsewhere that 30% of those with Medicaid coverage qualified through the Medically-Needy program. Thus we expect that many of the HIV+ population with Medicaid do not have coverage of HAART.

In addition, the differences in the effectiveness of private and public insurance might be explained by differences in the timing of initiation of coverage. Some private insurers may place limits on when it will cover HAART, but Medicaid limits can be quite severe. Many states place limits on how many prescriptions can be filled per month, and since HAART therapy alone averages 4.8 prescriptions, these can limit coverage for not only HAART but also drugs to treat opportunistic infections associated with advanced disease. Many of the drugs also required prior authorization that restricted use to advanced illness. The result is that privately insured patients are able to start treatment earlier in the disease than the publicly insured, and the latter often have no coverage at all. The results suggest that extending public insurance coverage to HIV+ patients in the early stage of the disease and creating incentives to increase treatment intensity and the use of HAART might prevent a significant number of premature HIV related deaths. These findings are especially relevant in light of the fact that treatment of HIV with prescription drugs not only saves lives but also result in lower total health care costs as

⁷Currently, 39 states offer drug coverage to the medically-needy.

increases in pharmaceutical costs are offset by reduction in hospitalization and other related costs.

References

- Bound, J., Jaeger, D., and Baker, R. (1995). Problems with instrumental variables estimation when the correlation between the instruments and the endogenous explanatory variable is weak. *Journal of the American Statistical Association*, 90(430):443–450.
- Bundorf, K. (2002). Employee demand for health insurance and employer health plan choices. *Journal of Health Economics*, 21(1):65–88.
- Cantor, C., Long, S., and Marquis, S. (1995). Private employment-based health insurance in ten states. *Health Affairs*, 14(2):199–211.
- Center for Disease Control (1998). Diagnosis and reporting of hiv and aids in states with integrated hiv and aids surveillance — united states, january 1994 - june 1997. *Morbidity and Mortality Weekly Report*, 47(15):309–314.
- Duan, N., McCaffrey, D. F., Frankel, M. R., St. Clair, P. A., Beckman, R., Keesey, J. W., Chien, C., Goldman, D. P., Smith, S., and Morton, S. C. (1999). Hcsus baseline methods technical report: Weighting, imputation and variance estimation. RAND Report MR-1060-AHCPR, RAND, Santa Monica, CA.
- Employee Benefit Research Institute (2001). Sources of health insurance and characteristics of the uninsured: Analysis of the march 2001 current population survey. EBRI Issue Brief #240.
- Fauci, A., Braunwald, E., Isselbacher, K., and Martin, J., editors (1998). *Harrison's Principles of Internal Medicine, 14th Edition*. New York: McGraw Hill.
- Fleishman, J., Hsia, D., and Hellinger, F. (1994). Correlates of medical service utilization among people with hiv infection. *Health Services Research*, 29(5):527–538.
- Goldman, D., Bhattacharya, J., McCaffrey, D., Duan, N., Leibowitz, A., Joyce, G., and Morton, S. (2001). The effect of insurance on mortality in an hiv+ population in care. *Journal of the American Statistical Association*, 96(405):883–894.

- Heckman, J. (2001). Micro data, heterogeneity, and the evaluation of public policy: Nobel lecture. *Journal of Political Economy*, 109:673–748.
- Horner, R., Bennett, C., Achenbach, C., Rodriguez, D., Adams, J., Gilman, S., Cohn, S., Dickinson, G., DeHovitz, J., and Weinstein, R. (1996). Predictors of resource utilization for hospitalized patients with pneumocystis carinii pneumonia (pcp): A summary of effects from the multi-city study of quality of pcp care. *Journal of Acquired Immune Deficiency Syndromes and Human Retrovirology*, 12:379–385.
- Institute of Medicine (2002). *Care Without Coverage: Too Little, Too Late*. Institute of Medicine.
- Joyce, G., Goldman, D., Leibowitz, A., Duan, N., Carlisle, D., Shapiro, M., and Bozzette, S. (1998). Variation in inpatient resource use in the treatment of hiv - do the privately insured receive more care? *Medical Care*, 37(3):220–227.
- Lancaster, T. and Intrator, O. (1998). Panel data with survival: Hospitalization of hiv-positive patients. *Journal of the American Statistical Association*, 93(441):46–53.
- Levy, H. and Meltzer, D. (2001). What do we really know about whether health insurance affects health? Working Paper #274, Joint Center for Poverty Research.
- Mroz, T. (1999). Discrete factor approximations in simultaneous equation models: Estimating the impact of a dummy endogenous variable on continuous outcomes. *Journal of Econometrics*, 92(2):233–274.
- Nelson, C. R. and Startz, R. (1990). Some further results on the exact small sample properties of the instrumental variable estimator. *Econometrica*, 58(4):967–976.
- Nyman, J. (1999). The value of health insurance: The access motive. *Journal of Health Economics*, 18(2):141–152.
- Palella, F., Delaney, K., Moorman, A., Loveless, M., Fuhrer, J., Satten, G., Aschman, D., Holmberg, S., and The HIV Outpatient Study Investigators (1998). Declining morbidity and mortality among patients with

advanced human immunodeficiency virus infection. *New England Journal of Medicine*, 338(13):853–60.

Shapiro, M. F., Morton, S. C., McCaffrey, D. F., Andersen, R. M., Berk, M. L., Bing, E. G., Cleary, P. D., Cunningham, W. E., Fleishman, J. A., Kanouse, D. E., Senterfitt, J. W., and Bozzette, S. A. (1999). Variations in the care of hiv-infected adults in the united states. results from the hiv cost and services utilization study. *Journal of the American Medical Association*, 281:2305–2315.

Staiger, D. and Stock, J. H. (1997). Instrumental variables regression with weak instruments. *Econometrica*, 65(3):557–586.

Table 1: Insurance, Use of HAART, and AIDS

Insurance status	% of Sample	HAART by first followup (%)	AIDS (%)
None	18	32	21
Public	52	34	53
Medicaid Only	28	33	47
Medicaid and Medicare	16	33	58
Medicaid and Private	2	42	57
Medicare Only	4	43	64
Medicare and Private	2	53	62
Private (only)	30	50	34

Source: HCSUS first follow-up (N=2466).

Table 2: Medicare beneficiary 1-year mortality—logit regression

Variables	Odds Ratio	95% Conf. Interval	
		Lower Limit	Upper Limit
State Instruments			
Medically-Needy Threshold	1.001	0.997	1.004
AFDC Threshold	1.001	0.997	1.003
SSI Threshold < 65% of FPL	1.140	0.816	1.592
Average Firm Size	0.985	0.924	1.050
Number of ADL limitations			
No Limitation (Ref. cat.)	-	-	-
1 ADL	2.375	1.803	3.129
2 ADL	2.503	1.819	3.443
3 ADL	3.296	2.307	4.723
4 ADL	3.376	2.331	4.889
5 ADL	5.857	4.347	7.893
6 ADL	7.589	5.786	9.954
Self reported Health Status			
Excellent (Ref. Cat)	-	-	-
Very Good	1.625	1.042	2.536
Good	1.839	1.205	2.805
Fair	2.415	1.574	3.703
Poor	4.283	2.7726	6.618

Source: Medicare Current Beneficiary Survey, 1996 (N=9,968). Excludes states not included in the HCSUS sample.

Table 3: Descriptive Statistics

Variable	Proportion or mean (N = 2,466)
Insurance Status	
Private Insurance	30%
Public Insurance	52%
Uninsured	18%
Demographics	
Age	39 years
Nonwhite	51%
Female	23%
Education	
Less than HS degree	25%
High school degree	27%
Some College or more	48%
Years since diagnosis:	
Less than one year	9%
1-2 years	12%
2-3 years	23%
3-4 years	27%
4-5 years	18%
5+ years	11%
State Instruments	
Medically-Needy Threshold	50% of FPL
AFDC Threshold	181% of FPL
SSI Threshold < 65% of FPL	7%
Average Firm Size	147 workers
Lowest ever CD4 count (cells per mm^3)	
500+	8%
200 – 499	38%
50 – 200	31%
0 – 50	23%
Died within 12 months after interview	4%

Note: FPL – Federal Poverty Line;

Source: HCSUS first followup survey (N=2,466)

Table 4: Results for the naïve model

Coefficient	No severity controls	With severity controls
Insurance		
Private	-0.0442	-0.3071
Public	0.4521**	0.1389
No Insurance (Ref. Cat.)	-	-
Demographics		
Age	0.0139	-0.0098
Age ²	-0.0001	0.0003
Nonwhite	-0.1327	-0.1520
Female	-0.0516*	0.0154
Education		
High school degree	-0.0134	-0.0075
Some College or more	0.0326	-0.0568
Less than HS degree (Ref. Cat.)	-	-
Years since diagnosis		
Less than one year	0.1836	0.0633
1-2 years	0.2441	0.3820
2-3 years	-0.0200	-0.0152
3-4 years	0.3746**	0.4657**
4-5 years	0.0761	0.1171
5+ years (Ref. Cat.)	-	-
Lowest ever CD4 count (cells per mm^3)		
500+	-	-0.9305**
200 – 499	-	-1.3007**
50 – 200	-	-0.7420**
0 – 50 (Ref. Cat.)	-	-
Constant	-2.5688**	-1.3810

Note: Estimates are from a single equation (weighted) probit of one-year mortality with and without severity controls (N=2,466).

** Statistically significant at 95% confidence level

* Statistically significant at 90% confidence level

Table 5: Mortality Results (Deaths per 1,000 HIV+ patients)

	Insurance Status		
	Private	Public	None
Unadjusted Raw Means	19	63	26
Naïve Model			
No severity controls	20 (-9%)	59 (+168%)	22
With severity controls	21 (-46%)	51 (+31%)	39
Structural Model	25 (-79%)	41 (-66%)	120

Note: Numbers in parentheses report percentage change in mortality relative to no insurance case (RI) for each empirical model (equations 13 and 14).

Table 6: Structural Model—Mortality Equation

Parameters	Estimate
Insurance	
Private	-6.7153***#
Public	-5.2764***#
No Insurance (Ref. Cat.)	-
Demographics	
Age	-0.2059
Age ²	0.0032
Nonwhite	-0.5536
Female	0.3652
Education	
High school degree	0.4813
Some College or more	-0.5750
Less than High School (Ref. Cat.)	-
Years since diagnosis	
Less than one year	-1.9967*
1-2 years	-0.1727
2-3 years	-0.9995
3-4 years	-0.1966
4-5 years	-0.2927
5+ years (Ref. Cat.)	-
Lowest ever CD4 count	
500+ cells per mm^3	-4.5941**
200 – 499 cells per mm^3	-4.8722**
50 – 200 cells per mm^3	-3.5580**
0 – 50 cells per mm^3 (Ref. Cat.)	-
Constant	-0.6411
Correlations	
$Corr(\rho_m, \rho_{private})$	0.325
$Corr(\rho_m, \rho_{public})$	0.892
$Corr(\rho_{public}, \rho_{private})$	0.717

Note: Estimates are maximum likelihood estimates of equation (12) (N=2,466). Correlations are computed using equations (8)-(10).

** Statistically significant at 95% confidence level

* Statistically significant at 90% confidence level

Coefficient on private insurance is not equal to coefficient on public insurance at 95% confidence level

Table 7: Structural Model—Insurance Equations

Parameters	Private Insurance	Public Insurance
Demographics		
Age	0.0932*	-0.0143
Age ²	-0.0007	0.0007
Nonwhite	-0.9247**	0.0508
Female	-0.0141	0.7064**
Education		
High school degree	0.8768**	-0.2089
Some College or more	1.5504**	-0.6675**
Less than High School (Ref. Cat.)	-	-
Years since diagnosis		
Less than one year	-1.0090**	-1.4389**
1-2 years	-0.0787	-0.5448**
2-3 years	-0.1152	-0.4162
3-4 years	0.0215	-0.0699
4-5 years	0.2848	0.2300
5+ years (Ref. Cat.)	-	-
Lowest ever CD4 count		
500+ cells per mm^3	-0.8453**	-1.8698**
200 – 499 cells per mm^3	-1.1291**	-1.8803**
50 – 200 cells per mm^3	-1.0054**	-1.1391**
0 – 50 cells per mm^3 (Ref. Cat.)	-	-
State-Level Instruments		
SSI Threshold < 65	-0.1179	-0.1526
AFDC Threshold	-0.0107*	-0.0142**
Medically-Needy Threshold	0.0167**	0.0166**
Average Firm Size	0.1494**	0.0461
Constant	-2.7945	3.6412
Wald test (of instruments)	41.83**	38.08**

Note: Estimates are maximum likelihood estimates of equation (12) (N=2,466). Correlations are computed using equations (8)-(10).

** Statistically significant at 95% confidence level

* Statistically significant at 90% confidence level

Coefficient on private insurance is not equal to coefficient on public insurance at 95% confidence level.